L6 ANSWER 2 OF 4 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2001271079 MEDLINE
DOCUMENT NUMBER: 21199564 PubMed ID: 11303031
TITLE: **Transgenic** studies of **pain** and analgesia: mutation or background genotype?.
AUTHOR: Lariviere W R; Chesler E J; Mogil J S
CORPORATE SOURCE: Department of Psychology and Neuroscience Program, University of Illinois at Urbana-Champaign, Champaign, Illinois 61820, USA.
CONTRACT NUMBER: DA 11394 (NIDA)
DA12735 (NIDA)
SOURCE: JOURNAL OF PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS, (2001 May) 297 (2) 467-73. Ref: 45
Journal code: 0376362. ISSN: 0022-3565.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
RS1. J6
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010529
Last Updated on STN: 20010529
Entered Medline: 20010521

AB The application of **transgenic** (knockout) technology to the study of **pain** is rapidly expanding. Despite its power, this technique has several shortcomings that complicate the interpretation of the data obtained. Although compensation by other genes is a well recognized problem, issues related to the background genotype of the mutant **mice** are less well appreciated. This review describes these confounds as they apply to studies of **pain** and **pain** inhibition. We show that the 129 and C57BL/6 **mouse** strains, which provide the default **genetic background** on which null mutants are constructed, display significant and sometimes extreme phenotypic differences in many assays of nociception, hypersensitivity, and analgesia. Although problems related to the differential responsiveness of the two strains are minimized by placing knockouts onto "pure" 129 and/or C57BL/6 backgrounds, we also illustrate that neither of these strains are particularly representative of inbred **mice** in general. Procedures to reduce confounds and converging evidence must be used to accurately determine the functions of the targeted genes in **pain**-related phenomena.

L6 ANSWER 3 OF 4 MEDLINE on STN
ACCESSION NUMBER: 1998016424 MEDLINE
DOCUMENT NUMBER: 98016424 PubMed ID: 9354804
TITLE: Disruption of the **mouse** L1 gene leads to malformations of the nervous system.
AUTHOR: Dahme M; Bartsch U; Martini R; Anliker B; Schachner M; Mantei N
CORPORATE SOURCE: Department of Neurobiology, Swiss Federal Institute of Technology, ETH-Honggerberg, Zurich, Switzerland.
SOURCE: NATURE GENETICS, (1997 Nov) 17 (3) 346-9.
Journal code: 9216904. ISSN: 1061-4036.
QH431. N363
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199712
ENTRY DATE: Entered STN: 19980109
Last Updated on STN: 19990129
Entered Medline: 19971204

AB The adhesion molecule L1 is a member of the immunoglobulin superfamily. L1 is involved in various recognition processes in the CNS and PNS, and

binding to L1 can activate signal transduction pathways. Mutations in the human L1 gene are associated with a variable phenotype, including mental retardation and anomalous development of the nervous system, referred to as 'CRASH' (corpus callosum hypoplasia, retardation, adducted thumbs, spastic paraplegia, and hydrocephalus). We generated an animal model of these conditions by gene targetting. Mutant **mice** were smaller than wild-type and were less sensitive to touch and **pain**, and their hind-legs appeared weak and uncoordinated. The size of the corticospinal tract was reduced and, depending on **genetic background**, the lateral ventricles were often enlarged. Non-myelinating Schwann cells formed processes not associated with axons and showed reduced association with axons. In vitro, neurite outgrowth on an L1 substrate and fasciculation were impaired. The mutant **mouse** described here will help to elucidate the functions of L1 in the nervous system and how these depend on genetic influences.

L8 ANSWER 6 OF 7 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 1999219413 MEDLINE
DOCUMENT NUMBER: 99219413 PubMed ID: 10204719
TITLE: Heritability of nociception I: responses of 11 inbred **mouse** strains on 12 measures of nociception.
AUTHOR: Mogil J S; Wilson S G; Bon K; Lee S E; Chung K; Raber P; Pieper J O; Hain H S; Belknap J K; Hubert L; Elmer G I; Chung J M; Devor M
CORPORATE SOURCE: Department of Psychology, University of Illinois at Urbana-Champaign, Champaign 61820, USA..
jmogil@s.psych.uiuc.edu
CONTRACT NUMBER: DA11394 (NIDA)
DA11888 (NIDA)
NS35057 (NINDS)
+
SOURCE: PAIN, (1999 Mar) 80 (1-2) 67-82.
Journal code: 7508686. ISSN: 0304-3959.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990714
Last Updated on STN: 19990714
Entered Medline: 19990630

AB It is generally acknowledged that humans display highly variable sensitivity to **pain**, including variable responses to identical injuries or pathologies. The possible contribution of **genetic** factors has, however, been largely overlooked. An emerging rodent literature documents the importance of genotype in mediating basal nociceptive sensitivity, in establishing a predisposition to neuropathic **pain** following neural injury, and in determining sensitivity to pharmacological agents and endogenous antinociception. One clear finding from these studies is that the effect of genotype is at least partially specific to the nociceptive assay being considered. In this report we begin to systematically describe and characterize **genetic** variability of nociception in a mammalian species, Mus musculus. We tested 11 readily-available inbred **mouse** strains (129/J, A/J, AKR/J, BALB/cJ, C3H/HeJ, C57BL/6J, C58/J, CBA/J, DBA/2J, RIIIS/J and SM/J) using 12 common measures of nociception. These included assays for thermal nociception (**hot plate**, Hargreaves' test, tail withdrawal), mechanical nociception (von Frey filaments), chemical nociception (abdominal constriction, carrageenan, formalin), and neuropathic **pain** (autotomy, Chung model peripheral nerve injury). We demonstrate the existence of clear strain differences in each assay, with 1.2 to 54-fold ranges of sensitivity. All nociceptive assays display moderate-to-high heritability (h2 = 0.30-0.76) and mediation by a limited number of apparent **genetic** loci. Data comparing inbred strains have considerable utility as a tool for understanding the genetics of nociception, and a particular relevance to **transgenic** studies.

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=> s l4 and hot plate
L7          17 L4 AND HOT PLATE

=> dup rem
ENTER L# LIST OR (END):l7
PROCESSING COMPLETED FOR L7
L8             7 DUP REM L7 (10 DUPLICATES REMOVED)

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